

MINNESOTA DEPARTMENT OF NATURAL RESOURCES
DIVISION OF FISH AND WILDLIFE
ECOLOGICAL SERVICES SECTION

LEAD RESIDUES IN CANADA GEESE
COLLECTED FROM LAC QUI PARLE WMA
IN 1978-79

Game Investigational Report No. 9

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August 1979

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By

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Background

Large numbers of crippled geese and a sizable migratory population of Bald Eagles at Lac Qui Parle Wildlife Management Area led Department of Natural Resources Biologists to suspect that these eagles might be exposed to toxic levels of lead through the ingestion of body shot geese. As a result, the Wildlife Section of the DNR proposed and developed a plan to analyze both geese and eagles for lead. Approximately 300 geese were collected for analysis during the 1978-79 Lac Qui Parle post-season dead waterfowl search. These geese were initially analyzed by X-ray for evidence of lead shot. This analysis revealed that a high percentage of the dead geese contained lead shot in the gizzard. A number of these geese also exhibited physical signs of chronic lead poisoning (Table II). Since most of the goose hunting at Lac Qui Parle WMA occurs on upland sites, lead poisoning was not previously suspected as a major impact to the waterfowl resource. An evaluation of the X-ray analysis prompted the DNR to propose an in-depth study of lead residues in the geese collected at Lac Qui Parle WMA. Such information would be valuable in assessing future management plans and the possible shift to the use of steel shot.

The Ecological Services Chemistry Laboratory agreed to analyze 250 to 300 goose liver tissues for lead residues pending funding and time constraints. Funds for the analysis were not directly available from the Federal Fish and Wildlife Service. However, the FWS agreed to analyze 50 of the geese for lead at their laboratory in Columbia, Mo. The Ecological Services Section of the DNR agreed to provide the funding for the analysis of the remaining tissue samples. Since this analysis was unanticipated it necessitated the hiring of one analyst by emergency appointment for the maximum allowed period of 30 days. These actions provided the Chemistry Laboratory with sufficient man hours and funds to analyze the liver tissues presented in this report without interrupting customary laboratory services for the DNR.

Methods

Liver tissues were dissected from whole goose samples, placed in 18 oz. whirl Pak[®] Bags, labeled and stored in the laboratory freezer until analysis. A .6 to .8 gram section of tissue was dissected from the frozen liver and placed in a 25 ml Parr[®] teflon lined digestion bomb and weighed. Three ml of Ultrex[®] brand nitric acid was added to the bomb which was then sealed and placed in a 150° C oven for 2 hours. The resulting digestate was rinsed into a 25 ml volumetric flask and brought up to volume with deionized distilled water. This analyte was stored in acid washed 60 ml polyethylene bottles at 5 ° C pending analysis.

The samples were analyzed by flame atomic absorption spectrophotometry on a Perkin-Elmer model 603 spectrophotometer. Preliminary analysis of duck liver tissues (Table III) indicated that it was not necessary to use the more sensitive but more time consuming flameless analysis to separate poisoned and non-poisoned geese. All samples were compared to freshly prepared blanks and standards at the same acid concentration. Additionally, two tissue samples were spiked with known quantities of lead to evaluate method recovery (Table IV). Each sample was analyzed twice to confirm experimental findings.

The physical manifestations of lead poisoning in waterfowl can include severe emaciation, crop and/or proventricular impaction, bile staining of the gastrointestinal tract, and paralysis of wings or legs. These characteristics are good circumstantial evidence for the suspicion of lead poisoning, especially when more than one is observed, but they are not absolutely diagnostic.

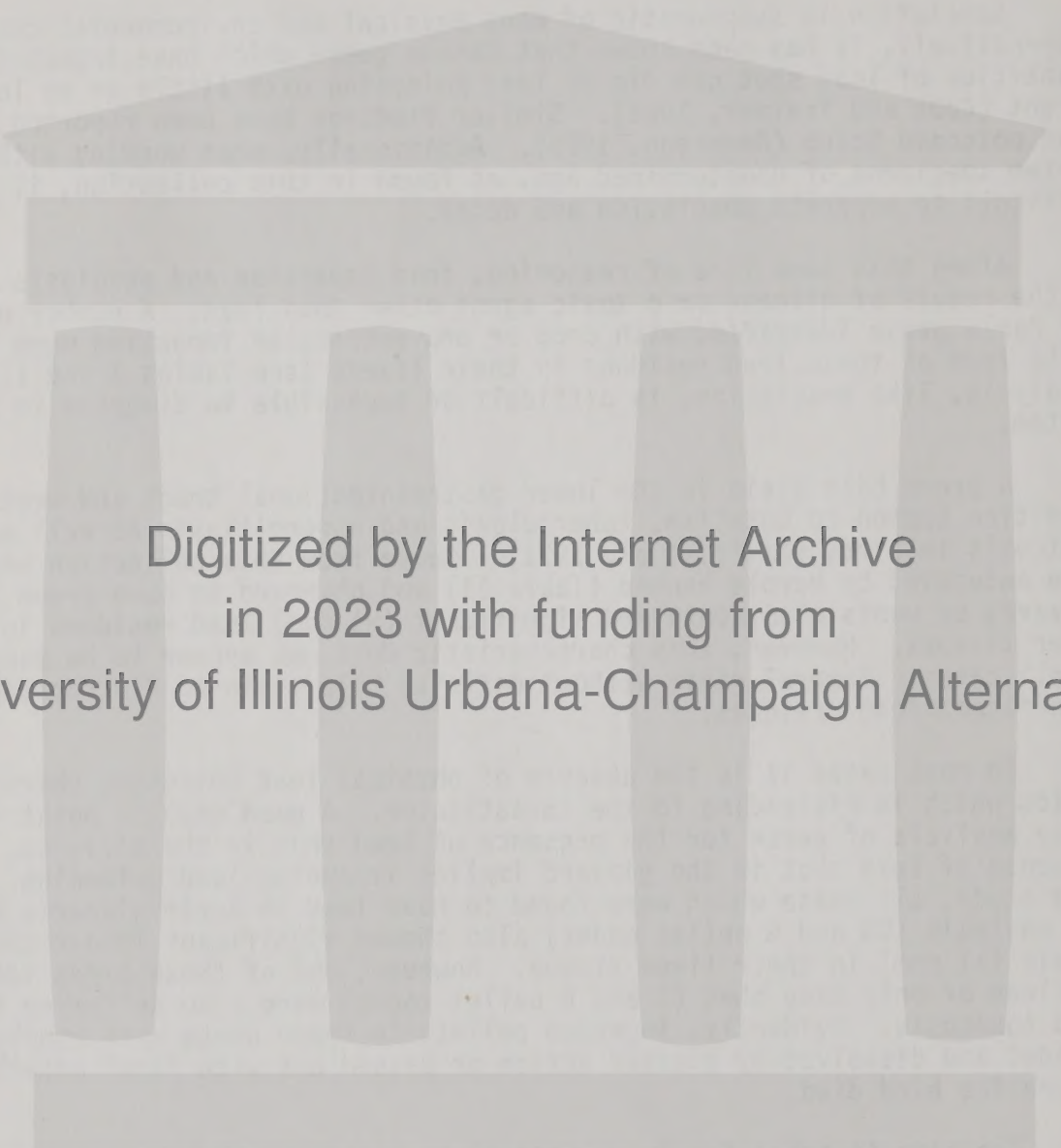
Emaciation is symptomatic of many physical and environmental conditions. Alternatively, it has been shown that Canada geese which have ingested large quantities of lead shot can die of lead poisoning with little or no loss of weight (Cook and Trainer, 1966). Similar findings have been reported for lead poisoned Scaup (Anderson, 1975). Additionally, when working with post-mortem specimens of undetermined age, as found in this collection, it is difficult to separate emaciation and decay.

Along this same line of reasoning, food impaction and paralysis could be the result of disease or a toxic agent other than lead. A number of Lac Qui Parle geese identified with crop or proventricular impaction were determined to be free of toxic lead residues in their livers (see Tables I and II). Paralysis, like emaciation, is difficult or impossible to diagnose in post-mortem.

A green bile stain in the lower gastrointestinal tract and vent is a condition common to botulism, tuberculosis and aspergillosis as well as lead toxicosis in birds (Davis et al, 1971). Geese from this collection which were autopsied by Harold Hanson (Table II) and observed to have green stained gizzards or vents also contained significant (>1 ppm) lead residues in their liver tissues. However, this characteristic does not appear to be manifested in all cases. Several geese without definite bile staining did contain significant lead residues.

In most cases it is the absence of physical lead poisoning characteristics which is misleading to the investigator. A good case in point is the X-ray analysis of geese for the presence of lead shot in the gizzards. The presence of lead shot in the gizzard implies impending lead poisoning. In this study, all geese which were found to have lead in their gizzards by X-ray analysis (BG and G pellet codes) also showed significant lead residue levels (>1 ppm) in their liver tissue. However, 35% of those birds identified as clean or only body shot (C and B pellet codes) were also suffering from lead toxicosis. Evidently, ingested pellets in these geese were completely abraded and dissolved by gizzard action or passed out with fecal material before the bird died.

The time it takes for lead residues to build in the tissues to a lethal level is highly variable and may take from several days to several weeks depending on the physical condition and diet of the bird, environmental conditions, and the number of pellets ingested. In fact, the majority of birds suffering from lead toxicosis probably succumb to disease, predation and starvation long before actually dying from direct lead poisoning (Jacobson et al, 1977). Hunt (1960) found that 84.4% of the ducks which contained lead shot had only one shot in each gizzard. Jordan and Bellrose (1951) indicated



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that the immediate cause of death among ducks ingesting one lead shot was starvation. It is presumed that lead poisoning and death occurred over relatively long periods, possibly months, for many of the geese in the Lac Qui Parle collection and that the onset of harsh weather in late November and early December finally made these debilitated birds succumb. The interpretation is further handicapped by the fact that 54% of all geese with pellets in their gizzards were also body shot. The role that the body shot played in the stress or death of each goose is only a matter of speculation.

While physical characteristics provide a good field check, the most accurate diagnosis of lead poisoning is attained through direct analysis of body tissues for lead residues. Most birds contain some measureable quantity of lead in their body tissues since lead is a regularly occurring constituent of the plants and animals in their diet (Bagley and Locke, 1967). Longcore et al (1974) found that lead concentrations of 6 to 20 ppm in the kidneys and liver of mallard ducks indicated acute exposure to lead. The results of this analysis compare favorably with this and other studies from the literature and with those samples analyzed for this study by the U. S. Fish and Wildlife Service (unpublished). In this study, 81% of all the geese diagnosed as lead poisoned have liver residue concentrations between 6 and 30 ppm (Figure I). Only 7 of 166 geese with measureable liver residue levels contained less than 6 ppm lead. A small number of geese with intermediate residue levels were expected from a sample of this size. These examples probably represent developing cases of lead poisoning which expired from other causes. This is reinforced by the fact that 6 of these 7 geese were also body shot. Apparently, the difference between poisoned geese (set as >6 ppm for discussion sake) and non-poisoned geese (set as <1 ppm or the detection capability of this technique) is distinct and readily identified. The distribution of lead residues in the 166 lead-positive geese from this sample reveals a sharp increase in the number of geese with residues from 6 to 16 ppm (Figure I). Assuming that lead was at least a contributing factor in the deaths of these geese, this is the concentration range over which lead toxicosis appears to first become an important mortality factor. The range in measureable residue levels, 1.4 to 66.0 ppm, in the Lac Qui Parle collection exemplifies the variability in individual tolerances to lead under any given set of conditions. Considering these variables, it would be difficult to establish a threshold of lethal lead residue. From a management standpoint, any debilitating level of lead toxicosis would have to be considered eventually lethal. In short, with the larger sizes of shot used in goose hunting a single ingested pellet is potentially lethal.

Conclusions

Based on this sample, it appears that a majority of the geese found dead or dying during the 1978-79 post-season waterfowl search at Lac Qui Parle WMA suffered from lead toxicosis and/or its complications. One hundred sixty-six or 66% of the 254 geese analyzed were found to be at some stage of lead toxicosis. The prime source of lead pellets is assumed to

be upland feeding and grazing sites in traditional hunting areas. If this is the case, proper management techniques could be used to alleviate or control the situation. The short residence time of the main migrating flock and the upland source of lead pellets facilitate management. By eventually shifting to the use of steel shot and deep plowing those areas of high pellet concentration it might be possible to greatly reduce the incidence of lead pellet ingestion and thus reduce the potential for acute lead poisoning.

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APPENDIX

Table I - Results of the Lead Analysis of 254 Geese from Lac Qui Parle
WMA (ug/gm wet wt. liver tissue).

Sample No.	Pellet* Code	Lead Conc. ± 1.0	Sample No.	Pellet* Code	Lead Conc. ± 1.0
1	B	42.5	34	B	<1.0
2	C	<1.0	35	B	8.9
3	B	<1.0	36	B	<1.0
4	C	16.1	37	C	<1.0
6	G	21.5	39	C	<1.0
7	C	<1.0	40	B	<1.0
8	G	31.0	42	G	1.7
9	B	<1.0	43	B	11.0
10	C	<1.0	44	B	<1.0
11	C	<1.0	45	B	<1.0
12	C	<1.0	46	C	23.6
14	C	<1.0	47	C	<1.0
15	C	<1.0	48	C	<1.0
16	C	<1.0	49	B	<1.0
17	C	<1.0	50	C	<1.0
18	C	<1.0	51	G	45.5
20	B	12.1	52	C	<1.0
21	C	<1.0	53	B	<1.0
22	C	<1.0	54	G	25.6
23	BG	24.8	55	B	<1.0
24	C	<1.0	56	B	<1.0
26	BG	13.2	57	C	<1.0
27	C	<1.0	58	C	<1.0
28	C	<1.0	60	B	<1.0
29	B	<1.0	61	B	2.1
30	B	<1.0	62	B	<1.0
31	G	28.1	65	B	<1.0
33	B	<1.0	66	B	<1.0

Table I (Continued)

Sample No.	Pellet* Code	Lead +1.0 Conc. -1.0	Sample No.	Pellet* Code	Lead +1.0 Conc. -1.0
67	B	<1.0	105	B	20.6
68	C	<1.0	106	BG	25.9
69	B	<1.0	107	G	16.2
70	B	<1.0	108	BG	58.2
71	B	<1.0	109	C	21.6
72	B	1.35	110	G	15.7
74	C	<1.0	111	BG	29.4
75	B	<1.0	112	G	14.7
76	C	<1.0	113	G	14.3
77	C	<1.0	114	B	10.5
78	C	<1.0	115	G	32.4
80	BG	5.1	116	BG	22.2
81	B	<1.0	117	C	<1.0
82	C	<1.0	118	B	26.2
83	B	<1.0	119	G	13.5
85	B	<1.0	120	C	15.9
86	C	<1.0	121	G	25.9
87	BG	29.8	122	G	17.7
89	G	31.7	123	G	19.7
90	BG	9.1	124	BG	29.1
91	B	3.8	125	BG	13.3
92	BG	23.9	127	BG	16.4
94	G	59.4	128	G	13.2
95	BG	9.3	129	B	<1.0
99	G	23.3	130	C	<1.0
100	C	<1.0	132	BG	11.9
102	BG	41.6	135	B	<1.0
103	G	28.5	136	B	10.3

Table I (Continued)

Sample No.	Pellet* Code	Lead +1.0 Conc. -	Sample No.	Pellet* Code	Lead +1.0 Conc. -
137	G	12.2	167	BG	10.2
138	BG	44.7	168	B	14.5
139	G	10.7	169	G	19.7
140	G	31.5	170	G	29.6
141	G	21.3	171	BG	24.4
142	BG	14.9	172	G	10.6
143	G	54.9	173	B	<1.0
144	BG	23.2	175	BG	30.0
145	BG	13.0	176	BG	11.0
146	BG	11.1	177	C	14.7
147	BG	18.4	178	BG	36.5
148	C	7.9	179	C	17.1
149	BG	13.0	181	C	<1.0
150	G	28.8	184	B	28.4
151	G	22.7	185	C	<1.0
152	B	21.0	186	BG	20.5
154	BG	16.6	187	BG	19.1
155	BG	14.6	188	B	21.8
156	B	<1.0	189	C	11.3
157	BG	14.3	190	C	23.6
158	G	23.7	191	B	<1.0
159	G	24.9	193	BG	27.2
161	B	7.0	194	G	48.2
162	BG	29.6	195	BG	16.7
163	B	<1.0	198	BG	33.7
164	B	14.7	199	C	<1.0
165	C	14.8	200	G	28.4
166	G	25.3	203	B	17.7

Table I (Continued)

Sample No.	Pellet* Code	Lead + Conc. -1.0	Sample No.	Pellet* Code	Lead + Conc. -1.0
204	BG	24.9	234	B	1.8
205	BG	14.8	235	G	66.0
206	G	18.6	236	B	20.2
207	B	35.8	237	C	13.9
208	B	13.1	238	BG	20.9
209	G	32.5	239	B	37.9
210	G	27.6	240	BG	16.0
211	C	<1.0	242	BG	26.1
212	BG	9.9	243	BG	37.3
213	G	37.6	244	G	9.9
214	G	27.4	245	B	<1.0
215	G	22.6	247	B	<1.0
216	G	10.4	248	B	34.5
217	C	21.7	249	C	<1.0
218	BG	10.3	250	BG	23.5
219	B	<1.0	252	BG	31.1
220	G	12.4	253	C	20.1
221	C	<1.0	254	B	8.8
222	G	13.4	255	C	<1.0
223	BG	39.4	256	B	<1.0
224	BG	20.5	257	BG	25.2
225	C	<1.0	258	C	<1.0
228	B	<1.0	259	BG	27.1
229	BG	20.7	260	B	<1.0
230	B	<1.0	262	B	<1.0
231	B	12.2	263	C	<1.0
232	G	19.4	264	B	28.9
233	BG	16.8	265	G	31.3

Table I (Continued)

Sample No.	Pellet* Code	Lead Conc.	+1.0 -1.0	Sample No.	Pellet* Code	Lead Conc.	+1.0 -1.0
267	G	37.3		288	C	<1.0	
268	C	18.4		289	C	18.5	
270	BG	23.3		291	B	<1.0	
271	G	26.1		292	G	22.5	
272	BG	16.2		294	BG	14.8	
273	B	<1.0		295	C	<1.0	
276	BG	16.7		296	BG	13.0	
277	B	17.1		298	C	7.4	
280	G	28.5		299	B	1.71	
281	G	22.7		300	BG	22.6	
283	B	<1.0		301	BG	20.0	
284	G	21.4		302	BG	13.8	
285	BG	17.7		304	G	10.0	
286	B	13.4		305	C	10.9	
287	BG	19.2		306	BG	24.1	

* Goose sample, Pellet Code

C - Clean under X-ray, no pellets

B - Body shot only

G - Gizzard shot only

BG - Shot in body and gizzard

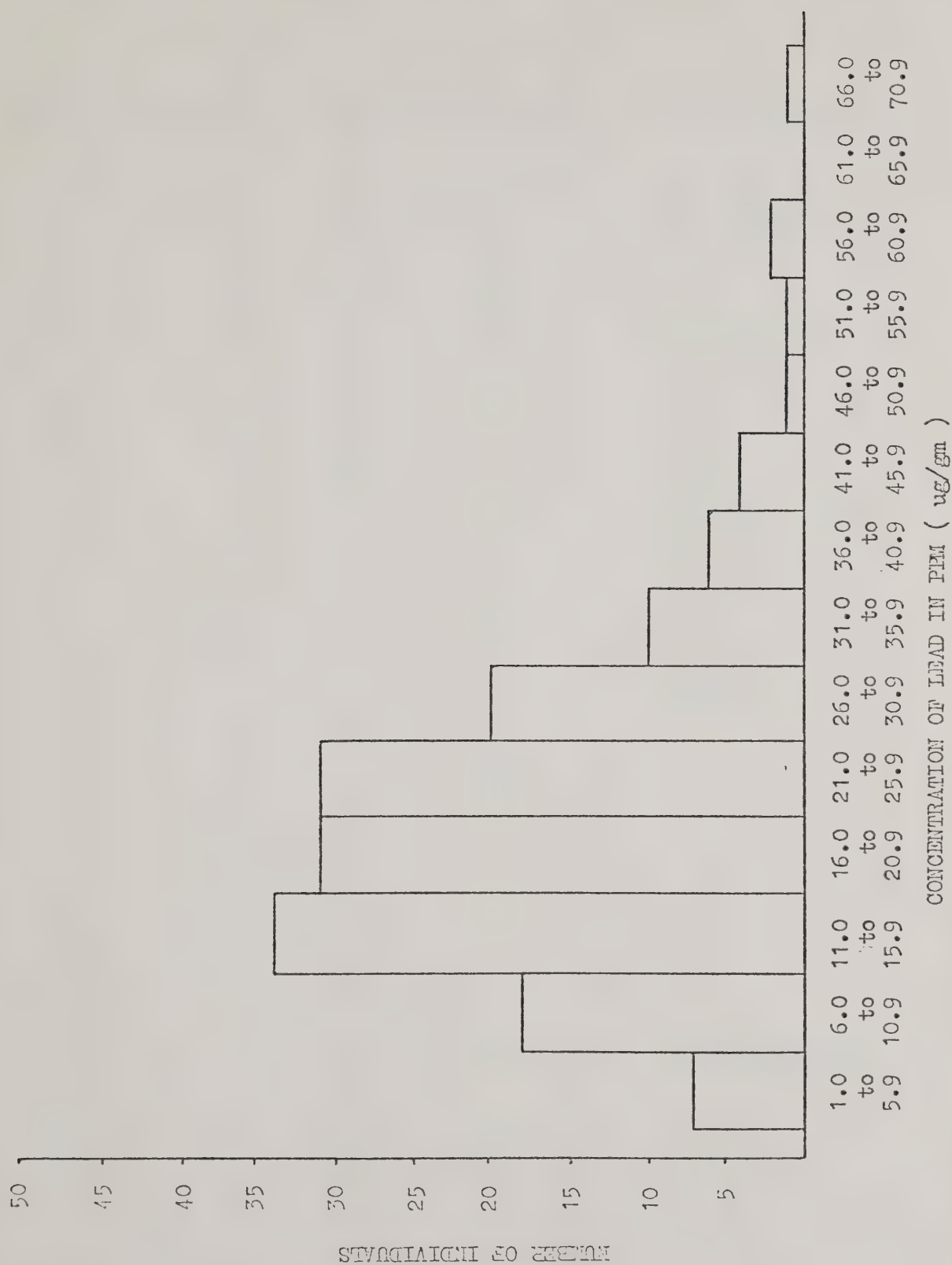


Figure I. The Distribution of Lead Residue Concentrations in the 166 Lead Positive Geese from the 1978-79 Lac Qui Parle Waterfowl Collection.

Table II - Physical post-mortem observations of 34 geese collected at Lac Qui Parle WMA as made by Harold Hanson, Illinois National History Survey.

Sample No.	Sex	Age	Weight(gms)	Observations
57	M	AD	--	--
65	M	AD	1700	V. thin
66	F	AD	--	Crop impacted Ex. thin
70	F	AD	2820	--
75	F	AD	1760	Crop impacted V. thin
83	M	YR	1500	Thin, bound crop
86	F	YR	--	--
91	F	YR	--	--
92	F	AD	3180	Gizzard contents V. green
103	F	YR	--	Shot in Pectoral, V. thin
111	F	YR	2640	Green vent
118	F	AD	--	Impacted provent Green vent
120	F	AD	--	--
123	F	AD	2800	--
124	F	AD	2800	--
128	F	YR	--	--
130	F	AD	--	V. thin, impacted crop
145	F	AD	3020	Normal
148	F	YR	--	--
164	F	YR	--	--
177	F	AD	2800	Normal condition Gizzard dark green
181	F	AD	--	--
184	M	AD	3600	Green vent
188	F	AD	--	--

Table II - (Continued)

Sample No.	Sex	Age	Weight(gms)	Observations
189	F	IM	2400	--
198	M	AD	--	--
200	F	YR	--	--
210	F	AD	--	--
232	F	IM	--	--
248	F	YR	1580	V. thin
250	F	AD	--	--
252	F	AD	--	--
264	F	AD	--	Bound proventriculus
277	F	YR	--	V. thin

Table III - Results of the lead analysis of 12 mallard ducks from
Lac Qui Parle WMA (ug/gm wet wt. liver tissue)

<u>Sample No.</u>	<u>Lead Conc. ± 1.0</u>
M-1	38.8
M-2	23.3
M-12	16.8
M-35	21.4
M-39	<1.0
M-51	1.1
M-54	17.0
M-61	19.1
M-62	24.4
M-86	22.3
M-87	20.4
M-111	70.7

Table IV - Recovery of lead from digested standards and spiked samples (ug/gm)

Sample	<u>Conc. in Digestate</u>		% Recovery
	Expected Conc.	Recorded Conc.	
M-62 + 2ppm	2.45 (2+.45)	2.45	100%
M-86 + .2ppm	0.68 (.48+.20)	0.69	101%



3 0112 077692306

Table 1 - Recovery of lead from spiked samples (ug/g)

Sample	Recovery Conc.	Recovery Conc.	Recovery %
W-52 + 20ppm	2.46 (2.40)	2.46	100%
W-52 + 20ppm	0.58 (0.50)	0.57	98%